

Current controversies

Action control is not affected by spatial neglect: A comment on Coulthard et al.

M. Himmelbach^{a,*}, H.-O. Karnath^a, M.-T. Perenin^{b,c}

^a Section Neuropsychology, Center for Neurology, Hertie-Institute for Clinical Brain Research, University of Tübingen, Hoppe-Seyler-Str. 3, 72076 Tübingen, Germany

^b Department of Integrative Neurosciences, INSERM U846, Stem Cell and Brain Research Institute, 18 Avenue Doyen Lépine, 69675 Bron Cedex, France

^c Université Claude Bernard Lyon I, Lyon, France

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Coulthard, Parton, and Husain (2006) recently published an elaborated and noteworthy review on deficits of motor control in patients with spatial neglect. Based on their survey and the presentation of two single cases with optic ataxia, they presented detailed anatomical conclusions on the neural implementation of action control in humans that challenged recent behavioural and anatomical findings.

Their line of reasoning was based on the discoveries of temporal and spatial motor deficits in stroke patients suffering from spatial neglect. Several studies claimed that such patients take more time to initiate a new movement and/or demonstrate longer movement times when these movements are directed towards the side contralateral of the brain lesion (Heilman, Bowers, Coslett, Whelan, & Watson, 1985; Husain, Mattingley, Rorden, Kennard, & Driver, 2000; Mattingley, Bradshaw, & Phillips, 1992; Mattingley, Husain, Rorden, Kennard, & Driver, 1998). Other studies revealed spatial inaccuracies. Patients with spatial neglect either demonstrated a terminal pointing error or an increased curvature or variability of trajectories (Goodale, Milner, Jakobson, & Carey, 1990; Harvey, Milner, & Roberts, 1994; Jackson, Newport, Husain, Harvey, & Hindle, 2000). However, a closer inspection of the experimental design of those studies reveals that these motor abnormalities in neglect patients resulted from direct comparisons between brain-damaged patients with spatial neglect and healthy controls without brain damage only (Goodale et al., 1990; Harvey et al., 1994; Heilman et al., 1985; Husain et al., 2000; Jackson et

al., 2000; Mattingley et al., 1992, 1998). Based on these studies, we thus can reason about the presence of certain deficits in patients with a circumscribed brain lesion. However, this does not necessarily lead to the conclusion that these deficits represent a specific impairment caused by the presence of spatial neglect. To investigate the specific influence of spatial neglect on motor functions, a comparison of neglect patients with patients suffering from brain damage but not spatial neglect is required (i.e. not only a comparison between neglect patients and healthy controls). Interestingly, studies incorporating such a group of brain-damaged control patients did not reveal any specific deficit in neglect patients neither in the spatial (Harvey et al., 2001; Himmelbach & Karnath, 2003; Karnath, Dick, & Konczak, 1997) nor in the temporal (Harvey et al., 2001; Konczak & Karnath, 1998) domain of goal-directed motor performance. Himmelbach and Karnath (2003) investigated the performance of stroke patients with acute neglect, patients who recovered from neglect, stroke patients with right brain damage but no neglect, and healthy subjects in a pointing task. Unfortunately, Coulthard et al. (2006) summarised these results stating that “. . . neglect patients did have greater absolute curvature . . .”. Presumably, they referred to the only significant difference in action control that was found, namely a difference between brain-damaged patients with neglect and healthy controls. As reviewed above, this has been observed in several previous studies. However, the decisive comparisons between brain-damaged patients with and without neglect did not reveal any significant differences (Himmelbach & Karnath, 2003). This result was congruent with another comprehensive group study of reaching movements in brain-damaged patients with and without neglect (Harvey et al., 2001). In contrast to the interpretation given by Coulthard et al. (2006), one would thus have to summarise the

* Corresponding author at: Section Neuropsychology, Center of Neurology, University of Tübingen, Hoppe-Seyler-Str. 3, D-72076 Tübingen, Germany. Tel.: +49 7071 29 84080; fax: +49 7071 29 5957.

E-mail address: marc.himmelbach@uni-tuebingen.de (M. Himmelbach).

currently available data on this issue by concluding that spatial and temporal abnormalities in reaching movements do not seem to be a consequence of spatial neglect but rather indicate a phenomenon occurring in some of these patients as well as in other stroke patients (without neglect), i.e. a phenomenon occurring with (so far not further identified) brain damage.

In their recent review article, Coulthard et al. (2006) further undertook an attempt to integrate findings from neglect patients and patients with optic ataxia. They refer to the recent lesion mapping study on optic ataxia published by Karnath and Perenin (2005) that compared lesion locations in 16 patients with optic ataxia and 36 patients without optic ataxia. Regrettably, Coulthard et al. (2006) summarised the findings by stating that “. . . the inferior parietal lobe is actually a critical region associated with optic ataxia”. In doing so, they focused on only one subregion of the centre of lesion overlap observed in that study. In fact, Karnath and Perenin (2005) found optic ataxia associated with a lesion overlap that affected the lateral and medial parieto-occipital junction (POJ) in both hemispheres. At the lateral convexity the centre of lesion overlap affected the junction between the inferior parietal lobule (IPL), superior parietal lobule (SPL), and the superior occipital cortex. The area of lesion overlap further extended via the underlying white matter towards the medial cortical aspect of the hemisphere and included the precuneus close to the parieto-occipital sulcus. These findings fit surprisingly well with a recent event-related fMRI study in healthy subjects (Prado et al., 2005). The authors measured the brain activity when participants reached either towards a target represented on the fovea or towards an extrafoveal target. The analysis revealed increased signals bilaterally at the POJ for targets in the peripheral visual field, i.e. exactly for the situation in which brain-damaged patients with optic ataxia show misreaching. Optic ataxia patients demonstrate dramatic visuo-motor impairments if their movements are directed to targets in the peripheral visual field. As soon as they fixate the target their impairments vanish or decrease considerably.

We agree with Perenin (1997) as well as Coulthard et al. (2006) that patients with optic ataxia and patients with spatial neglect show double dissociable deficits. Optic ataxia patients show deficits in reaching for a target under peripheral vision but no abnormalities in exploration tasks that require tactile hand movements covering a large area in peripersonal space (Perenin, 1997). Vice versa, patients with neglect demonstrate a marked ipsilesional shift of exploratory movements while their goal-directed movements are essentially normal. The cortical regions associated with the two disorders obviously have different functional roles (Perenin, 1997). Coulthard et al. (2006) addressed this anatomo-functional issue by presenting two single cases with optic ataxia. Their case GS had a circumscribed lesion affecting the right precuneus and SPL. The other case BN demonstrated a much larger lesion affecting wide parts of the medial aspects of the right parietal cortex and underlying white matter. A comparison of these individual lesions with the contrast templates that derived from the quantitative group analysis of 16 patients with optic ataxia by Karnath and Perenin (2005) shows that GS's lesion is located right within the centre of critical lesion overlap identified by Karnath and Perenin (2005) (see

Figs. 4 and 5 of that study). Also, the much larger lesion of BN covered the medially located aspect of this centre of overlap, beyond further areas.

While Coulthard et al. (2006) emphasised the similarities between their two individual cases, the comparison between GS and BN also revealed marked anatomical differences. The latter is not surprising since lesions of individual patients rarely are restricted to a well-specified anatomical site but include various regions related and not related to the disorder. To identify those areas that are typically associated with a certain impairment (optic ataxia in the present case), i.e. that are affected in a representative sample of patients with this disorder, it is indispensable to study a larger group of patients showing the same disturbance. However, even if such a group of patients has been investigated, a simple overlap of their lesions does not discriminate between regions that are commonly damaged (e.g. due to the vasculature of these regions) and regions that have a role in the function being investigated (e.g. optic ataxia). Therefore, an anatomical comparison with a control group of patients who also suffered a brain lesion but do not show the pathological behaviour is needed (Rorden & Karnath, 2004). Anatomical conclusions drawn without such a comparison at the group level or by presenting only single cases with individual brain lesions may simply reflect vulnerability to vascular injury without close specificity for the function of interest or may reflect only part of the structures that are integral to this function.

Given (i) the lack of motor control deficits in reaching movements that are specific for neglect patients (i.e. deficits that are observed only in patients with neglect but not in other stroke patients without neglect), and (ii) that vice versa patients with optic ataxia show a specific disturbance of this function, we suggest that there are two dissociable underlying cognitive functions which can be separately disrupted by distinct brain lesions. The lateral and medial aspects of the POJ appear to be involved in the fast control of visually guided reaching, on the basis of short-lived and implicit eye- or arm-centred space representations, as part of the dorsal action system. In contrast, more ventral regions involving the temporo-parietal junction (TPJ), the superior temporal cortex and insula – damaged in patients with spatial neglect (Heilman, Watson, Valenstein, & Damasio, 1983; Karnath, Fruhmann Berger, Küker, & Rorden, 2004; Mort et al., 2003) – seem to play an essential role in adjusting body position relative to external space (Karnath & Dieterich, 2006). These areas appear to subserve longer lasting, explicit and multimodal spatial representations.

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